

Type-D Personality and Heart Disease: It Might Be ‘One Small Step’, but It Is Still Moving Forward: A Comment on Grande et al.

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Since the landmark studies of the late 60s and early 70s, we as a field have tried to find the key psychosocial predictors of poorer cardiovascular outcomes. In the mid-90s, a new construct, type-D personality, was found to have significant predictive ability for mortality in patients with coronary heart disease [1]. This has led to a constant stream of original studies and reviews exploring the potential role of type-D personality in the progression of heart disease. Of particular note, there has been a recent spate of systematic reviews [2–4] on the topic. In spite of this, the review by Grande et al. [5] truly provides an extension, not only through the inclusion of new studies, but also in its conceptualisation. Grande and colleagues have taken a rigorous and refined approach to assessing the impact of type-D personality on cardiac outcomes, which has created a number of interesting talking points.

One particularly interesting point rightly highlighted by the authors is the contrasting data on patients with coronary artery

disease vs. those with congestive heart failure (CHF). They raise the issue of whether this difference is driven by a true lack of prognostic effects in CHF patients or if this just reflects the increased sample size in the newer larger CHF studies and tend, with good reason, to lean more on the side of an actual prognostic difference. There is, however, an alternative possibility which revolves around the appropriate cut point for different populations. The type-D scale, which was used in 11 out of the 12 studies, was developed in a specific population, a Flemish/Dutch coronary artery disease population [6], and whilst there has been some good work to establish the factor structure invariance of the scale across different languages and populations [7], the same cannot be said for the cut point of 10. One of the main underpinnings of good psychometrics is that all aspects of a scale or questionnaire are validated when used in new populations, i.e. any population that differs substantially from the original cohort the scale/questionnaire was developed in. Following this, it could be that type-D personality may be predictive of outcomes in CHF populations, but not with a cut point of 10. Further complicating this issue is the way in which the original cut point was generated. The figure of 10 is derived from a median split of negative affect and social inhibition in the original validation study. We think all of us would argue that this is not the optimal way of defining a diagnostic level, though, as highlighted by Grande and colleagues, what is the comparator or ‘reference standard’ [8] needed to determine the classification accuracy of the type-D cut point? Suffice to say, there needs to be more work around the optimisation of a type-D cut point, or if no valid measure can be reliably generated, then only continuous data should be used in the future.

Another interesting finding from this meta-analysis was that no matter what way the data were analysed, the pooled odds ratio or hazard ratio (HR) ranged from 1.5 to 2.3, though for those sub-analyses with less studies, the increased confidence intervals suggested non-significance. At this point in time, it

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would seem that type-D personality is a relatively robust predictor of outcomes and seems to be comparable to other psychosocial measures such as depression [9] and anxiety [10]. To reiterate the point raised by the authors, where does this leave us in understanding the complex collinear associations between all these measures and heart disease? Of note, the sub-analysis which only included the six studies that adjusted for other psychosocial measures found the adjusted HR was 1.83 (95% CI [0.99–3.37]). Some would argue that this is good evidence of a truly independent effect of type-D personality, due to the comparable effect size to the main analyses and the closeness of the lower CI to 1 in light of the limited number of studies and sample size. In contrast, others would argue that this is good evidence to support no independent effect of type-D due to the non-significant finding. Most probably, this result only provides us with the impetus to further explore this issue of collinearity with large, well defined populations and validated questionnaires (including appropriate cut points) and ultimately identify the toxic psychosocial ‘phenotype’. Given that it is highly unlikely that there will be consensus on this kind of data collection in the future, it will be important for future meta-analyses to estimate these effects using higher level statistical approaches, such as Bayesian hierarchical models [11] or marginal structural models [12], and adjust analyses with objective methodology quality scales.

One quirky aspect of the review that struck us, being Europeans who are now in North America, was the fact that all the included studies were derived from European populations. Extending the points raised above, will there be an appreciable difference if European populations are systematically compared against North American populations? If there is, how much would methodological issues confound such a finding? Only time will tell.

Finally, the review and meta-analysis by Grande et al. does provide us with an additional perspective on the role of type-D personality in heart disease; though this is probably only ‘one small step’, it does provide some solid indications on where the literature needs to go so that we can take the ‘giant leap’ forward in ultimately defining the toxic psychosocial ‘phenotype’ for heart disease development and progression.

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